requirement of a1KAP-dependent subcellular targeting and PKD2-dependent Ca^{2+} signals for activation of KV CaMK-II. CaMK-II activation offers a mechanistic link between left-sided KV Ca^{2+} elevation and the appearance of LPM *spaw*. Supported by National Science Foundation IOS-0817658.

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Program/Abstract # 322
c21orf59 regulates cilia motility and acts through the PCP pathway

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Program/Abstract # 323
Characterization of a novel mouse mutant *schlei* with Sonic Hedgehog signaling and cilia defects

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An ENU-mutagenesis screen uncovered a recessive mouse mutant, *schlei*, with preaxial polydactyly and variable loss of the floor plate in the neural tube. These and other partially penetrant defects suggest an association with the Sonic Hedgehog (Shh) signaling pathway. Additionally, *schlei* mutants are characterized by disrupted formation of primary cilia, which have been shown to be critical for Shh responsiveness. Ventral cell types of the neural tube are lost or reduced in *schlei*, demonstrating a defect in high-level Shh activation. Conversely, *schlei* mutant limbs are characterized by ectopic activation of downstream targets of Shh signaling, suggesting a loss of Gli3 repressor function. Preliminary epistasis studies demonstrate that the *schlei* mutation partially rescues ventral neural tube patterning in *shh* mutants, lending further support to the hypothesis that the *schlei* gene product acts within the Shh pathway. Interestingly, cilia formation appears to be affected in only a subset of ciliated tissues in *schlei*, suggesting the possibility of specific regional requirements for the *schlei* gene product. Future work will focus on mapping the mutation to a genetic locus, as well as further characterizing the mutant and the role the gene plays in the Shh pathway.

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Program/Abstract # 324
AKAP11 modulates the neural tube patterning through the direct binding and the phosphorylation of Patched1 and Smoothened

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Hedgehog receptor Pathed1 (Ptc1) and its transducer Smoothened (Smo) control dorso-ventral patterning and cell survival of neural tissue. These receptors localize and function in primary cilia and the basal body, however, how they control these specific processes is not fully understood. Here we report that A kinase anchoring protein 11 (AKAP11) is expressed in the basal body and can directly bind to and regulate the phosphorylation of Ptc1 and Smo. Furthermore, we show that AKAP11 knockdown in the neural tube alters dorso-ventral patterning and affects cell survival. AKAP inhibition leads to constitutive accumulation of Ptc1 and Smo in primary cilia and activation of PKA. Thus, AKAP11 governs neural tube patterning and cell survival by controlling the activity of Ptc1 and Smo in primary cilia and the basal body through PKA.

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Program/Abstract # 325
Fat-Hippo signaling regulates the proliferation and differentiation of *Drosophila* optic neuroepithelia

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The *Drosophila* optic lobe develops from neuroepithelial progenitor cells; we identify here a role for the Fat–Hippo pathway in controlling its growth and differentiation. Mutation of tumor suppressor genes within the pathway, or expression of activated Yorkie, promotes overgrowth of neuroepithelial cells, and delays or blocks their differentiation; mutation of *yorkie* inhibits growth and accelerates differentiation. Neuroblasts and other neural cells, by contrast, appear unaffected. Neuroepithelial cells undergo a cell cycle arrest before converting to neuroblasts; this is regulated by Fat–Hippo signaling. Combinations of cell cycle regulators, including E2f1 and Cyclin D, can delay neuroepithelial progression, and Fat–Hippo signaling delays differentiation in part through E2f1. We also identify a role for Notch signaling, and an influence of Fat–Hippo signaling on expression of the Notch ligand Delta. Our studies establish that Fat–Hippo signaling regulates the proliferation of neuroepithelial cells, and identify transient cell cycle arrest and upregulation of Delta as crucial for the progression of neuroepithelial cells to neuroblasts.

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Program/Abstract # 326
The effects of perturbing components of the Notch signaling pathway on neurotransmitter phenotype and calcium channel subunit expression in *X. laevis*

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Many defects and abnormalities affecting the brain and spinal cord arise from the incorrect neurotransmitter phenotype specification of neurons during development. Given the role of Notch signaling in neurogenesis, the Notch pathway is a possible mediator of this differentiation because of its role in lateral inhibition. We hypothesized that Notch signaling is involved in the specification decision between GABAergic and glutamatergic fates and that activating Notch *in vivo* would result in more neurons acquiring a glutamatergic neurotransmitter phenotype, while inactivating Notch would increase GABAergic phenotypes. To test this hypothesis, we activated Notch signaling by injecting mRNA for X-Notch ICD and inactivated Notch signaling by injecting mRNA for xSu(H) DNA Binding Mutant, an inactive form of the transcription factor xSu(H). Embryos injected with X-Notch ICD showed reduced expression of the glutamate transporter *xVGlut1* and the GABA transporter *xGAT1*, and embryos injected with xSu(H) DBM showed widespread ectopic expression of neuronal markers *xNBT* and *xVGlut1*. Calcium levels have a known impact on neurotransmitter phenotype specification; therefore, we have characterized how Notch affects the